Published in final edited form as:
Curr Opin Allergy Clin Immunol. 2009 April ; 9(2): 154–160.

Asthma and Ethnic Minorities: Socioeconomic Status and Beyond

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Abstract

Purpose of review—We aim to discuss current insights into our understanding of the mechanisms by which socioeconomic status (SES) influences the prevalence and severity of asthma in ethnic minorities. In addition, we review potential risk factors for ethnic disparities in asthma that are not mediated by SES.

Recent findings—Exposures and factors correlated with ethnicity through SES (e.g. indoor and outdoor air quality, smoke exposure, and access to healthcare) are likely to explain a significant proportion of the observed ethnic differences in asthma morbidity. However, other factors correlated with ethnicity (e.g., genetic variation) can impact ethnic disparities in asthma independently of and/or interacting with SES-related factors.

Summary—SES is a rough marker of a variety of environmental/behavioral exposures and a very important determinant of differences in asthma prevalence and severity among ethnic minorities in the U.S. However, SES is unlikely to be the sole explanation for ethnic disparities in asthma, which may also be due to differences in genetic variation and gene-by-environment interactions among ethnic groups.

Keywords

Childhood asthma; asthma epidemiology; socioeconomic status; ethnicity; race; minorities

Introduction

Asthma, a global public health problem [1], affects over 6.8 million children and adolescents in the U.S.[2]. There is profound variability in the prevalence and morbidity of asthma among ethnic groups [3].

Ethnicity is strongly correlated with socioeconomic status (SES) in the U.S., where members of certain ethnic groups (e.g., African Americans, Puerto Ricans) are disproportionately represented among the poor. Because poverty has been associated with increased asthma morbidity, it has been postulated that SES is solely responsible for ethnic differences in asthma and asthma morbidity. The effect of SES on illnesses such as asthma is likely

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mediated through pathways including environmental exposures, access to health care, stress, and psychological/cultural factors [4]. However, ethnicity is also correlated with racial ancestry, which may influence asthma disparities through differences in the frequency of disease-susceptibility alleles.

The purpose of this article is to review current evidence to support the role of SES and other factors as potential explanations for ethnic-related differences in asthma, and to suggest potential future directions for research in this field.

Asthma and Asthma Morbidity in Ethnic Minorities

The prevalence, morbidity, and severity of asthma are higher in children who belong to certain ethnic minorities [5, 6], and/or whose households report indicators consistent with low SES [7**, 8]. Although the overall prevalence of current childhood asthma in the U.S. is 8.7%, it varies widely by ethnicity, ranging from 4–5% in Asian Indians and Chinese to 19% for Puerto Ricans, with non-Hispanic whites and other minorities ranking in the middle [2, 3, 9*, 10*] (Table 1). Similarly, the rate of current asthma in children from families below the federal poverty threshold is higher (11.1%) than in families above it (7.7–8.5%)(10). Asthma severity is higher in certain ethnic groups such as Puerto Ricans and African Americans [11]. African Americans have more ER visits, hospitalizations, and higher mortality rates from asthma than whites [3]. In contrast to the low mortality rates from asthma among Mexican-Americans (0.3 per 100,000), mortality among Hispanics in New York City, which has a large proportion of Puerto Ricans, is approximately 1.3 per 100,000 [12**].

We will review the main mechanisms and potential factors underlying the association between socioeconomic status, ethnicity and asthma.

Environmental exposures

Many environmental factors influence the pathogenesis and severity of asthma:

Indoor allergens

Compared to rural areas and suburbs, indoor allergen levels are higher in urban households in low-income areas and in those hosting multiple families [13*, 14]. Inner-city households have higher levels of indoor allergens such as cockroach, which are associated with increased asthma morbidity. Differences in allergic sensitization among ethnic groups are more pronounced in inner-city environments. Using data from inner-city children in the Third National Health and Nutrition Examination Survey (NHANES III), Stevenson et al. found that Mexican Americans were three times more likely and that African Americans were four times more likely to be sensitized to cockroach than whites (after adjustment for age, gender, and indicators of SES factors)[14]. Children with asthma who live in the inner city also tend to have more ER visits for asthma than their counterparts from rural regions [15].

Residence in inner-city areas partly explains the high levels of exposure of certain ethnic minorities to high levels of indoor allergens [5]. Two studies of asthmatic children in the U.S. Northeast showed that Hispanic and African-American ethnicity are associated with reduced exposure to high levels of dust mite allergen but increased exposure to cockroach allergen, even after accounting for indicators of SES [16, 17]. Potential explanations for the observed association between ethnicity and indoor allergen exposure include residual confounding by housing characteristics and/or behavioral differences among ethnic groups. Although a nationwide survey showed no association between ethnicity and dust mite

allergen levels in the beds of U.S. homes, it was limited by small sample size and thus had inadequate statistical power [18].

Ethnicity has been associated with patterns of allergic sensitization (atopy) in children with and without asthma. African-American and Puerto Rican children (with and without asthma) are more likely to be sensitized to cockroach and dust mite than white children [14]. Since African Americans have been shown to be exposed to relatively low levels of dust mite allergen, this finding suggests ethnic differences in susceptibility to sensitization to specific allergens.

Although most children with asthma are atopic, a significant proportion of atopic children do not have asthma. This dissociation between atopy and asthma varies by ethnicity. For example, Mexican Americans have a similar prevalence of atopy but a lower prevalence of asthma than Puerto Ricans. Determinants of ethnic differences in susceptibility to asthma in atopic children have been largely unexplored.

Cigarette Smoking

Approximately 20% of U.S. adults smoke [19] with significant variation by SES: smoking prevalence is ~46% in people with a General Education Development (GED) diploma, 22% for those with a college education, and ~7% for persons with a graduate degree. Smoking is also more prevalent among subjects living below the federal poverty level (31%). Smoking rates vary widely among ethnic groups, with American Indians and Alaska Natives having the highest rates at ~32%, and Asians the lowest at 10%. Despite marked differences in asthma prevalence and morbidity, African Americans and whites have the same rates of cigarette smoking (approximately 22–23%). Among 12- to 17-year-olds participating in a survey from 1999 to 2001, reported smoking rates were 28% for American Indians / Alaska Natives, 16% for whites, 11% for Hispanics, and 7% for non-Hispanic blacks [20].

Pre- and post-natal exposures to cigarette smoking are associated with asthma and asthma morbidity in childhood [21*]. *In utero* smoke exposure varies widely among ethnic minorities: 20% in American Indians, 16% in whites, 10% in Puerto Ricans and non-Hispanic blacks, 5% in Japanese, 3% Mexicans, and 1.5% in Central / South Americans [22]. *In utero* smoke exposure also varies by insurance type and education status [23].

During childhood, the prevalence of tobacco smoke exposure and levels of salivary cotinine are higher in children with asthma symptoms and doctor-diagnosed asthma, with a more pronounced difference in children from lower SES [24*]. Smoke exposure increases asthma morbidity; conversely, smoke-free laws have been associated with fewer asthma ER visits both in children and in adults [25*].

Smoking behavior among adults varies with ethnicity and SES, with members of certain ethnic groups (e.g., Puerto Ricans) smoking more often and/or more heavily than members of other groups (e.g, Mexicans). Thus, differences in parental smoking could account for part of the observed ethnic disparities in childhood asthma. However, few studies have tried to assess the effects of smoking on ethnic differences in asthma. In a study of over nine thousand people, Beckett *et al.* found that an association between Hispanic origin (mainly Puerto Rican) and increased risk of asthma was not influenced by passive exposure to smoking at home [26].

Air pollution

Outdoor pollutants can trigger asthma exacerbations and may play a role in asthma pathogenesis. Non-whites are more likely to live in areas with elevated levels of air pollutants, including particulates, carbon monoxide, ozone and sulfur dioxide [27, 28**]. A

study in New York City showed higher rates of asthma exacerbations and hospitalizations in children from highly polluted areas such as the Bronx, which also has a high percentage of residents from minority populations [29].

Nitrogen oxide and diesel exhaust particles (DEP), markers of traffic-related air pollution, have also been associated with increased asthma symptoms [30**]. Recent data suggest that the effect of DEP may be modified by genetic polymorphisms: in a cohort of children in Cincinnati, high DEP exposure was associated with increased risk of wheezing only in carriers of allele Val(105) in the gene for glutathione s-transferase π (GSTP1)[31].

Access to healthcare

Access to healthcare is determined by several factors, which in turn influence asthma morbidity.

Household income and insurance status

In a study of over 100,000 children (the National Survey of Children's Health), Flores *et al*. found marked differences between ethnic groups with regard to full-time employment rates, household income, and insurance coverage and type [32**]. In that study, the prevalence of asthma was higher in ethnic groups with relatively low employment rates, income, and insurance coverage (Figure 1). In New York City, asthma "hotspots" correspond to areas with higher concentrations of ethnic minorities, low-income households, and public housing [33].

Lack of adequate health insurance has a negative impact on asthma management by imposing barriers to appropriate diagnosis and treatment [28]. Recent advances in both long-term and acute asthma management may exacerbate such inequality, as they would only be accessible to those with adequate insurance. It should be noted, however, that lack of access to healthcare is unlikely to be the sole explanation for ethnic differences in asthma outcomes. For example, Puerto Ricans (who are U.S. citizens) have greater morbidity from asthma than Mexican immigrants in spite of easier access to healthcare.

Stress and comorbidities

Exposure to stress/violence and co-existing illnesses such as obesity and depression may partly explain the ethnic differences in asthma that are mediated by SES.

Exposure to stress and violence

Long-term maternal stress in early life has been associated with increased risk of childhood asthma, independently of other factors such as low SES [34*]. Cohen *et al.* recently reported that physical or sexual abuse was associated with current asthma morbidity in a cross-sectional study of Puerto Rican children [35**]. Family structure also plays a role, with children living with a single mother at higher risk for inadequate management of and increased morbidity from asthma [36*]. Together with results from other recent studies [37*,38*,39], these findings suggest that exposure to stress and violence (which is more common in ethnic minorities) influences the pathogenesis and morbidity of asthma in childhood.

Obesity

Obesity has been associated with asthma in different populations [40, 41*]. Among asthmatics in the Childhood Asthma Management Program (CAMP), the proportion of

overweight was higher in blacks and Hispanics than in whites and in members of other races [42*].

The influence of obesity on asthma could be due to several factors, many of which are associated with SES (e.g., diet and exercise). However, it has been reported that increased adiposity in infancy is associated with recurrent wheeze later in childhood [43*]. This points towards other mechanisms, such as genetic factors and a general inflammatory state [44**], which could predispose to airway inflammation. Severe obesity further impairs airflow due to increased chest wall resistance. Patients with obstructive sleep apnea (OSA) and habitual snoring not only have obstructive problems but also tend to have increased airway inflammation at baseline [45]. Several adipokines (cytokines produced by adipose tissue) have been implicated in airway inflammation [46*].

Depression and anxiety

Depression and anxiety are more prevalent in youth of lower SES [47] and/or with limited education (48). Adolescents with asthma have nearly twice the risk of depressive or anxiety disorders as adolescents without asthma [49*]. In a large population-based birth cohort in Finland, depressive symptoms and emotional behavioral problems before 8 years of age were associated with asthma in early adulthood [50]. Whether preceding or accompanying asthma, there is a clear relationship between depression and increased symptom report, poor medication adherence, and increased school absenteeism [51*]. Finally, co-morbid depression/anxiety are under recognized and undertreated in youth with asthma [52].

Psychological and cultural factors

Parental psychological and cultural factors may affect childhood asthma in several ways. Parents of black and Hispanic children worry more about their child's asthma but have lower expectations for symptom control and functionality, more competing priorities, and more concerns about over-medication and medication dependency than white parents [53**]. They also tend to have worse compliance with preventive medications, even when insurance coverage is not an issue [54]. Similar results have been elicited in individuals of South Asian descent and in other minorities in the United Kingdom [55*]. Among adolescent asthmatics in the U.S., minority and low SES subjects were more likely to have an inaccurate perception of their asthma control, with an evident tendency toward under-perception of symptoms [56].

Physician's attitudes and perceptions also play a role. Among a large sample of adult asthmatics, African Americans were more likely to have their asthma severity underestimated by treating physicians, resulting in less inhaled steroids usage, and less instruction on exacerbation management [57*]. Similar findings have been reported with minorities in the Netherlands [58], where universal healthcare is available.

Beyond socioeconomic status

We have reviewed several mechanisms by which SES may influence asthma, particularly in ethnic minorities. However, factors correlated with SES are unlikely to explain all the variability in asthma prevalence, severity and mortality among ethnic groups [59].

- Non-Hispanic blacks have a higher prevalence of current asthma, exacerbations, and hospitalization rates than whites *even after adjusting* for several demographic and socioeconomic factors [5].

- Puerto Rican children have *higher* and Asian children *lower* asthma prevalence and hospitalization rates than whites, even after adjusting for sociodemographic variables [60].
- Mexican Americans have *lower* asthma prevalence than most other groups, yet tend to have incomes and insurance coverage similar to that of African Americans and Native Americans; they are also more likely than whites, and almost as likely as African Americans, to be sensitized to indoor aeroallergens [14].

All of the above findings could be explained by residual (unmeasured) confounding by factors related to SES (e.g., housing characteristics, exposure to stress and violence). However, in a survey of over 3,000 individuals in almost 1,000 homes in the same area in Brooklyn (NY), Ledogar *et al.* [59] found that ethnic differences in asthma prevalence (5% in Dominicans vs. 13% in Puerto Ricans) were not influenced by residence (cluster or building), education level, country of education, or household size. This is a thought-provoking study, as differences in asthma prevalence were present in ethnic groups sharing very similar environments and SES within a same geographic area.

A potential explanation for part of the observed ethnic disparities in asthma is genetic predisposition. The heritability of asthma has been reported to be between 36 and 79%, and several groups have identified genomic regions and/or genes potentially implicated in the pathogenesis and/or severity of asthma [61**]. Although some of these studies have included members of ethnic minorities (e.g., African Americans, Hispanics, and Asians) most have been conducted in non-Hispanic whites. As an example, results for studies of three candidate genes for asthma are listed in Table 2. While there are potential asthma-susceptibility genes, none has been consistently replicated across all major ethnic groups. Ongoing genome-wide association studies (GWAS) have identified and will continue to identify asthma-susceptibility genes. Ethnic differences in the effect of a disease-susceptibility gene were recently reported in a GWAS of diabetes mellitus type II [72*]: of relevance, the observed differences were likely due to variation in allelic frequencies by ethnicity, as well as potential interactions between genetic variants and unmeasured environmental factors. Thus, recent findings suggest that well-conducted GWAS of asthma in ethnic minorities (including examination of gene-by-gene and gene-by-environment interactions) should provide valuable insights into the causes of ethnic disparities in asthma.

Conclusions and future directions

Differences in SES among ethnic groups are likely to influence ethnic disparities in asthma morbidity through several mechanisms. An improved understanding of these SES-related pathways is essential and could lead to a reduction in current asthma disparities. However, the interactions between these factors are very complex and difficult to dissect and thus comprehensive policies that address SES disparities as a whole should help reduce the asthma burden in ethnic minorities.

Although vigorous efforts to address SES-related risk factors for asthma are essential and should continue, we must also recognize the importance of understanding the impact of genetic variation and its interaction with environmental exposures on asthma pathogenesis in ethnic minorities.

Acknowledgments

Support: This work was supported by grants HL04370, HL066289, HL079966, HL073373 and T32 HL07427 from the National Institutes of Health.

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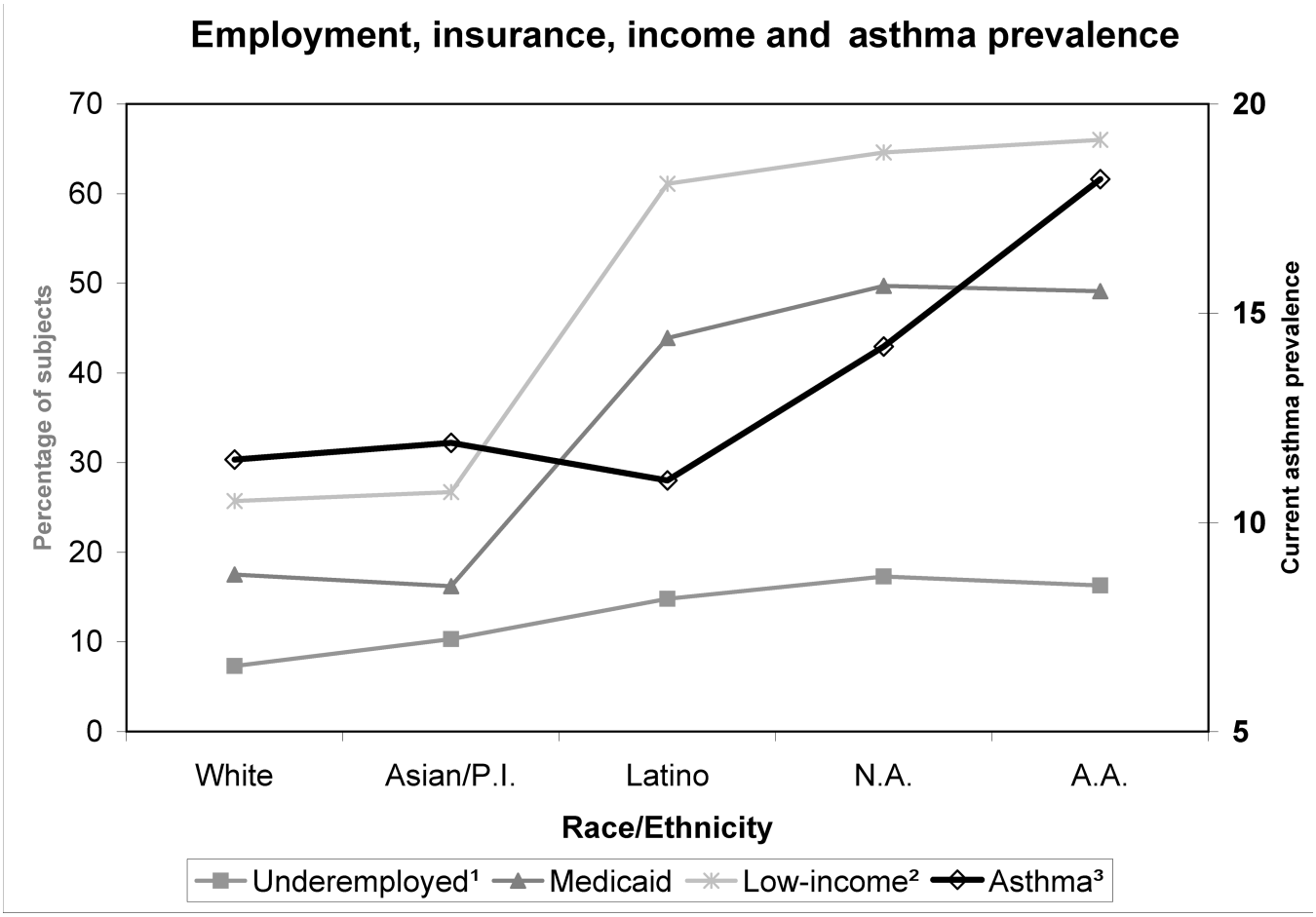

Figure 1. Underemployment, household income, insurance type, and asthma prevalence in children 0–17 years of age in the United States

Data from: Flores G, Tomany-Korman SC, *Pediatrics* 2008;121:e286-98[32]

[1]Percentage of households without a full-time employed adult. [2]Percentage of households with combined income below 200% of the federal poverty level. [3]Reported asthma prevalence. PI = Pacific Islander. NA = Native American. AA = African American.

Table 1

Asthma: current prevalence and mortality rates among children 0–17 years of age in the United States

Population	Current prevalence (%)	Mortality rate (per 1▩000▩000)
Puerto Ricans	19.2	*a*
AI/AN	13.0	*a*
Non-Hispanic blacks	12.7	9.2
Filipino	10.7	*a*
National average	9.3	2.6
Non-Hispanic whites	8.0	1.3
Mexicans	6.4	1.7
Chinese	5.1	*a*
Asian Indian	4.4	*a*

Data from MMWR 2007 [10•], ALA 2007 [2], CDC/NCHS 2006 [3], NHIS 2001–2005 [8]; Brim *et al.* [9•].

AI/AN, American-Indian/Alaska Native.

[a] No reliable data available.

Table 2

Selected candidate-gene association studies of asthma in different ethnic groups

Gene	Ethnic group	Results and comments
ADAM33	African-Americans	One study found three SNPs weakly associated with asthma or allergic sensitization [62]; another found no association [63].
	Hispanics	One study found two SNPs associated with asthma and allergic sensitization in Hispanics [62]; others found no association in Mexicans, Puerto Ricans, and Costa Ricans [63–65].
	Asians	One study found three SNPs associated with asthma and allergic rhinitis in Chinese [66]; another found no association in Koreans [67].
ADRB2	African-Americans	One study found no association of the Arg16Gly polymorphism with bronchodilator responsiveness [68].
	Hispanics	One study found an association with bronchodilator responsiveness in Puerto Ricans but not in Mexicans [69].
IL13	Hispanics	One study found an association with response to inhaled steroids and allergy-related phenotypes in Costa Ricans [70].
	Asians	One study found an association with response to leukotriene inhibitors in Koreans [71].

SNP, single-nucleotide polymorphism.